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LUNG FUNCTION, RESPIRATORY DISEASE, AND SMOKING IN FAMILIES[1,2]

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Schilling, R. S. F., A. D. Letai, S. L. Hui, G. J. Beck, J. B. Schoenberg and A. Bouhuys (Yale U. Lung Research Center, New Haven, CT 06510). Lung function, respiratory disease, and smoking in families. *Am J Epidemiol* 106: 274-283, 1977.

Respiratory symptoms, disease and lung function were studied in 376 families with 816 children who participated in a survey in three USA towns. Parental smoking had no effect on children's symptoms and lung function. Also, there was no evidence that passive smoking affected either lung function or symptoms of adults. There was no association between prevalence of self-reported cough and/or phlegm in parents and their children. There was a highly significant association between the prevalence of wheeze in parents and their younger children, for whom parents reported this symptom. Wheeze in children was also significantly associated with a parental history of asthma, and lung function was lower in children with a family history of asthma. Even after accounting for height, weight, age, sex and race, children's lung function correlated significantly with parents' lung function. However, the contribution of familial factors (i.e., parents' lung function, smoking, and history of asthma) to children's lung function is small compared to the effects of height, weight and age.

asthma; family characteristics; lung volume measurements; smoking

We investigated the contribution of familial factors, including smoking by parents, to lung function and respiratory symptoms in children. The study is a part of a population study in three towns in the United States in which members of families were questioned about respiratory symptoms and disease. Subjects were also given lung function tests measuring forced vital capacity (FVC), one second forced expiratory volume ($FEV_{1.0}$), peak expiratory flow rate (PEF) and maximum expiratory flow at 50 per cent of FVC (MEF50%) and at 25 per cent of FVC (MEF25%).

Children of smoking parents may suffer unduly from respiratory disease. For example, infants of mothers who smoke have significantly more attacks of bronchitis and pneumonia than infants of nonsmoking mothers (1). Also, children of smoking parents who may or may not have symptoms themselves have more bronchitis and pneumonia during their first year of life than children of nonsmoking parents (2), but this was not true during four subsequent years of follow-up. Nevertheless, older children may have an increased incidence of acute respiratory disease in homes where cigarettes are smoked (3, 4). These studies have led to the conclusion

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Abbreviations: $FEV_{1.0}$, forced expiratory volume in first second of forced expiration; FVC, forced expiratory vital capacity; MEF50% and MEF25%, instantaneous maximum expiratory flow rates at 50% and 25% of FVC, respectively, measured from recordings of maximum expiratory flow volume (MEFV) curves; PEF, peak expiratory flow rate.

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that passive smoking (inhaling other people's smoke) is injurious to health, particularly that of children (5).

The prevalence of cough in children however, is not only associated with parental smoking, but also with respiratory symptoms among parents (6, 7). After adjustment for the positive correlation between symptoms in parents and children, parental smoking had no significant effect on children's symptoms. Thus, smoking parents may affect their offspring's respiratory system indirectly, rather than via a direct effect of smoke. For example, smoking adults often cough and produce phlegm and thereby transmit infection (6).

All these results are based on children's symptoms as described by their parents. This may introduce bias. Parents who smoke have more symptoms and may be more conscious of them in their children. Parents who over- or under-report their own symptoms may do the same for their children. In this study, we have asked the children themselves some of the questions on respiratory symptoms. Also, children of smoking parents are more likely to smoke than children of nonsmokers (8). Unlike the previous studies, we have considered smoking habits of children in our analyses.

Familial resemblance in lung function, apart from the known factors of height, sex and ethnic group, appears to be slight, according to studies in twin pairs (9). Peak flow in five-year-old children correlates with peak flow in their parents (10), but this may be due to correlation between their heights. Peak flow in children was significantly related to their own histories of bronchitis and pneumonia, but there was no association between their peak flow and their parents' smoking habits, respiratory morbidity and social class. In the Tecumseh study (8), age- and height-adjusted $FEV_{1.0}$ scores correlated significantly not only between parents and their children, but also between siblings and even between spouses. However, these results were not controlled for smoking habits, nor were the possible effects of passive smoking on lung function considered. In the present study we have attempted to separate the effects of smoking from other familial factors on both lung function and respiratory symptoms.

Materials and Methods

This investigation was part of a large survey on lung disease in the three towns of Lebanon and Ansonia in Connecticut, and Winnsboro in South Carolina. Families in which both parents and at least one child between the ages of seven and 18 had been examined were identified by name, address and telephone number. Family groups were checked from replies by parents about the numbers and names of their children and replies by children about their brothers and sisters, to eliminate those in which there was any doubt about the accuracy of the family grouping. There were 418 families with data for both parents and 915 children. We only report on the 376 white families since there were too few blacks for detailed analysis. Table 1 shows the distribution of the white families by town and by age and sex of the children.

The methods used for interviewing subjects and for measuring lung function have been described elsewhere (11). Prediction equations for lung function in white males and females were derived from the data of all healthy, lifetime nonsmoking adults and children in the total survey population (12). From these equations, the proportion (R^2) of the total variance in lung function

Table 1

Distribution of families by town and by age and sex of children

	Lebanon	Ansonia	Winnsboro	Total
Families	247	54	75	376
Boys 7–17	244	50	59	353
Boys 18+	35	8	14	57
Girls 7–14	188	41	48	277
Girls 15+	83	18	28	129

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which is explained by height, weight, and age is determined.

The prediction equations were derived separately for boys aged 7–17 and men aged 18+, and for girls aged 7–14 and women aged 15+ (12). Since there were few older children, only the girls aged 7–14 and boys aged 7–17 (the age ranges in the prediction equations for children) were included in the present analysis of lung function. Residual lung function was obtained for each subject by subtracting the predicted value from the observed value. A residual of less than zero means that lung function is lower than expected from the data in healthy lifetime nonsmokers. Since the prediction equations for girls and boys use the natural logarithm (ln) of the function values, the residuals of children are obtained in transformed units (e.g., residual ln FVC); values for men and women are expressed in liters or liters/sec.

Symptoms and chest illnesses were defined as follows:

Cough and/or phlegm—usual cough at any time of the day or night, and/or usual phlegm production at any time of the day or night.

Wheeze—any history of wheezing or chest tightness.

Asthma—"yes" to the question, "Have you ever had bronchial asthma?"

Bronchitis—"yes" to the question, "Have you ever had bronchitis?"

Pneumonia—"yes" to the question, "Have you ever had pneumonia?"

Family history of asthma—"yes" to the question, "Have your parents, brothers, sisters, or children ever had asthma?"

All subjects including children answered the questions on smoking, cough and phlegm for themselves. Parents answered the questions on wheeze and on family history of asthma for children aged 15 and under. Data on chest illnesses in children were not analyzed because some of the answers were thought to be unreliable.

To relate the socioeconomic class of parents to the symptoms and lung function of their children, we classified fathers into eight groups based on the US Department of Labor's occupational classification of 1965 (13), and mothers as either housewives or those working outside their homes.

Results

Symptoms. We determined the prevalence of symptoms in groups according to symptom prevalence in parents: 1) neither parent had the symptom, 2) mother only had the symptom, 3) father only had the symptom, or 4) both parents had the symptom. Prevalences of cough and/or phlegm were highest in children in group 4, but there was no association between parents' and children's symptoms when all four groups were compared (table 2). The results were the same for children who had never smoked. For wheeze, the prevalence in parents correlated significantly with that in their children aged 15 years and under, but not with prevalence in older children, possibly because of smaller numbers (table 3). However, parents answered the wheeze questions for the younger children; only the older ones answered for themselves. Again, the results were similar in the subsample of children who had never smoked.

The association between parents' smok-

TABLE 2

Prevalence of cough and/or phlegm in children by cough and/or phlegm in their parents

Parents' cough/phlegm	All boys		All girls	
	Total	Cough/phlegm	Total	Cough/phlegm
		No (%)		No (%)
Neither	221	32 (14)	218	24 (11)
Mother	50	7 (14)	35	5 (14)
Father	113	19 (17)	105	10 (9)
Both	26	7 (27)	48	9 (19)
χ^2	2.91		3.06	
p	0.41		0.38	

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ing and children's symptoms was examined by comparing a group in which parents were both present nonsmokers with a group in which at least one parent was a present cigarette smoker. There were no significant differences between these groups in the prevalence of either cough and/or phlegm or wheeze in either boys or girls. When the nonsmokers were further divided into either ex-smokers or lifetime nonsmokers, the results remained insignificant. Also, there was no significant interaction between parent's symptoms and smoking in relation to children's symptoms.

TABLE 3

Prevalence of wheeze in children by age and by wheeze in their parents

Parents' wheeze	Total	Wheeze No. (%)	Total	Wheeze No. (%)
	Boys 7–15		*Boys 16+*	
Neither	154	22 (14)	66	10 (15)
Mother	44	10 (23)	23	4 (17)
Father	72	9 (13)	15	4 (27)
Both	29	13 (45)	7	3 (43)
χ^2	17.67		3.85	
p	<.001		0.28	
	Girls 7–15		*Girls 16+*	
Neither	172	16 (9)	59	9 (15)
Mother	49	10 (20)	16	4 (25)
Father	54	7 (13)	16	1 (6)
Both	33	10 (30)	7	0 (0)
χ^2	12.18		3.56	
p	<.01		0.31	

Although no association was found in the prevalence of cough and/or phlegm between mothers and fathers, there was a significant relationship in the prevalence of wheeze ($\chi^2 = 6.44$; $p < 0.025$). Symptoms in parents were related to their smoking habits. Cough, phlegm and wheeze were significantly more prevalent among male and female current smokers. However, prevalence of these symptoms in nonsmokers was unrelated to whether or not their spouses smoked.

We also attempted to relate symptom prevalences in children to their parents' reports of respiratory illness. Families were classified into non-mutually exclusive groups in which at least one parent ever had 1) bronchitis, 2) pneumonia, or 3) asthma. Each of these groups was compared to a group in which neither parent ever had any of these illnesses. There were no significant differences in the proportion of children with cough and/or phlegm in each parent-illness group, compared to the no-illness group. A significantly higher proportion of girls reported wheeze in the parents' asthma group; this was also true for boys in all three parent illness groups (table 4).

Symptom prevalences in children were similar among socioeconomic classes as defined by father's occupation. We also compared four groups defined by the mother's work and current smoking status (nonsmoking or smoking; housewife or working). Here, the only significant findings

TABLE 4

Prevalence of wheeze in children in relation to chest illnesses in their parent(s)

Parent(s)	All boys Total	All boys Wheeze No. (%)	All boys χ^2	All girls Total	All girls Wheeze No. (%)	All girls χ^2
No illness	161	17 (11)		170	19 (11)	
Bronchitis	137	36 (26)	11.45†	125	22 (18)	1.96
Pneumonia	173	35 (20)	5.22*	182	30 (16)	1.65
Asthma	41	14 (34)	12.24†	48	17 (35)	14.24†

* $p < .025$.

† $p < .001$ for comparisons between symptom prevalence in parent-illness group and parent no-illness group.

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was that daughters of smoking working women reported more wheeze than daughters of the other three groups ($\chi^2 = 10.5$; $p < 0.025$).

Lung function. To examine both active and passive smoking in relation to lung function in parents, we divided the families into four groups according to parents' current cigarette smoking: 1) both smokers, 2) father smoker, mother nonsmoker, 3) mother smoker, father nonsmoker, 4) both nonsmokers. Forty-six families in which one parent smoked only pipes or cigars were excluded because the numbers were too small. The results of an analysis of variance for residual $FEV_{1.0}$ in the four groups are shown in table 5. The highly significant differences in the mean residuals indicate the expected adverse effect of active smoking, with one exception: the men in the group where only their wives smoked also had low residuals. These men were further subdivided by their past smoking history. The men who never smoked themselves (but whose wives smoked) did not deviate significantly from normal; the male ex-smokers in the group had particularly low lung function. In the families in which only the father smoked, the mother's lung function was similar to that in which neither parent smoked. Thus, passive smoking was not an important factor affecting adult lung function.

To determine the effect of passive smoking on lung function in children, we used analysis of variance to compare lung function residuals of boys aged 7–17 or girls aged 7–14, divided according to the four parent smoking categories. Girls' MEF50% and MEF25% were lowest in those families in which only the mothers smoked, but differences were not significant. When the analysis was repeated for the subsample of children who had never smoked themselves, this difference for MEF50% was significant ($F = 2.65$; $p < 0.05$).

Residual lung function in children was also studied in relation to their parents' history of chest illness. Boys aged 7–17 and girls aged 7–14 were classified into parent-illness groups, as for the symptom analysis. For the asthma group, the five mean residuals were lower in both boys and girls than in the parent no-illness group. These differences were significant for $FEV_{1.0}$ ($t = 2.08$; $p < 0.05$) and PEF ($t = 3.51$; $p < 0.001$) in girls. The trend toward lower function residuals in children of parents with bronchitis or pneumonia was not significant.

From parents' answers to the questions on family history of asthma, we obtained information on the children's first and second degree relatives. All mean residuals were lower in the children who had at least one relative with asthma, compared to children who had none; results were significant for $FEV_{1.0}$, MEF50%, and MEF25% in boys (table 6).

There was no significant relation between family socioeconomic status and

TABLE 5

Mean residual $FEV_{1.0}$ of parents by current smoking category (n = no. of pairs)*

		Residual $FEV_{1.0}$, liters				
		Both smokers (n = 78)	Father smoker, mother nonsmoker (n = 74)	Mother smoker, father nonsmoker (n = 40)	Both nonsmokers (n = 136)	*F*
Fathers,	$\bar{x}$	−0.42	−0.35	−0.24	−0.06	11.66†
	(SEM)‡	(0.05)	(0.06)	(0.07)	(0.04)	
Mothers,	$\bar{x}$	−0.24	0.00	−0.13	−0.09	5.66*
	(SEM)	(0.04)	(0.04)	(0.07)	(0.03)	

* The results for residual FVC and flow rates show similar trends.

† $p < .01$.

‡ Standard error of mean.

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TABLE 6

Residual lung function in children in relation to family history of asthma*

		Boys 7–17			Girls 7–14		
		Family asthma (n = 158)	No family asthma (n = 195)	t	Family asthma (n = 119)	No family asthma (n = 158)	t
Residual $FEV_{1.0}$	x̄	−0.035	−0.002	2.57†	−0.020	0.004	1.85
	(SEM)	(0.009)	(0.009)		(0.010)	(0.009)	
Residual MEF50%	x̄	−0.044	0.001	2.64‡	−0.020	−0.010	0.56
	(SEM)	(0.012)	(0.011)		(0.013)	(0.011)	
Residual MEF25%	x̄	−0.055	0.000	2.85‡	−0.028	−0.017	0.54
	(SEM)	(0.013)	(0.014)		(0.015)	(0.013)	

* All children's lung function residuals are expressed on the natural logarithm scale, and not as liters or liters/sec.

† $p < 0.05$.

‡ $p < 0.01$.

TABLE 7

Correlations between residual lung function in spouses and in parent-child pairs (n = no. of pairs)

	Spouses (n = 376)	Father-son age 7–17‡ (n = 353)	Father-daughter age 7–14 (n = 277)	Mother-son age 7–17 (n = 353)	Mother-daughter age 7–14 (n = 277)
Residual FVC	.07	.19†	.21†	.15†	.12*
Residual $FEV_{1.0}$	.07	.17†	.23†	.11*	.16*
Residual PEF	.10	.19†	.20†	−.01	.06
Residual MEF50%	.07	.20†	.15*	.17†	.12*
Residual MEF25%	.07	.13*	.03	.26†	.09

* $p < .05$.

† $p < .01$.

‡ Age of child.

mean lung function residuals of sons or daughters of fathers from the various occupational groups. Also, there were no differences in children's residuals among the four groups defined according to mothers' work and smoking status (housewife or working; nonsmoking or smoking).

A comparison of lung function residuals between groups eliminates the effects of height, weight and age (12). Thus, any correlation between residuals of spouses, parents and children, or of siblings would suggest an effect of other factors, such as family resemblance in lung function (tables 7 and 8). Analysis of covariance showed that lung function residuals of children in the four parent smoking groups (both smokers; father smoker, mother nonsmoker; mother smoker, father nonsmoker; both nonsmokers) were the same after adjusting for mothers' and fathers' residuals. Hence, the different smoking groups may be combined for the analysis of parent-child correlations. In these correlations, each parent-child pair was counted independently; i.e., if a father had three sons, he was included three times, once with each son, in the father-son correlation. For the sibling correlations, the oldest sib was paired with the sib closest to him in age. Then the second oldest was paired with the third oldest and so on. Thus each sib except the oldest and youngest was a member of two pairs, once as the older and once as the youngest of the pair. None of the correlation coefficients between spouses were significant. In other pairs, at least three out of the five residuals yielded significant results, except between brothers. For children who had never smoked, the correlations were similar to those in all children shown in

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TABLE 8

Correlations between residual lung function in siblings (n = no. of pairs).

	Brothers age 7–17 (n = 103)	Sisters age 7–14 (n = 65)	Opposite-sex sibs (n = 214)	All sibs (n = 382)
Residual FVC	.15	.33†	.32†	.27†
Residual $FEV_{1.0}$	.12	.26*	.25†	.21†
Residual PEF	−.02	.26*	.16*	.12*
Residual MEF50%	.19	.09	.17*	.16*
Residual MEF25%	.04	.22	.13	.12*

* $p < .05$.
† $p < .01$.

tables 7 and 8. Positive correlations between parents' and children's lung function residuals persisted when families with histories of asthma in parents or relatives were excluded.

The contribution of familial factors and passive smoking to children's lung function can be assessed from a multiple regression anlaysis (table 9). The child's lung function measurement (not the residual) was the dependent variable. The independent variables were the height, weight, and age terms from the original prediction equation (12), and the parents' lung function residuals, the four parents' smoking categories, and a family history of asthma. Mothers' or fathers' residuals, or both, were significant variables for several measurements in boys as well as girls. The variable representing families in which the mother smokes was significant for MEF50% in girls. For girls aged 10 years, of average height and weight (148 cm, 37 kg), the predicted MEF50% was 2.86 liters/sec if the mother smoked, and 3.03 liters/sec if she did not. The family history of asthma variable was significant for boys' $FEV_{1.0}$ and MEF25%. Quantitatively, this variable, too, caused only a small difference in predicted values (e.g., at age 10, height 148 cm, weight 40 kg, $FEV_{1.0}$ for boys with a family history of asthma was 2.19 liters, versus 2.25 liters for those without). When only children who had never smoked were included in the analysis, the results were similar, except that the family history of asthma variable was also significant for boys' MEF50%.

Table 9 also shows the percentage of the variance (R^2) in the children's lung function measurements which is explained by the height, weight and age terms, and by the addition of the significant family terms. The greatest proportion of the variance is explained by the height, weight, and age terms (R_A^2). Relatively little is added by the parents' residuals, the smoking term, or the asthma term (compare R_F^2 with R_A^2).

DISCUSSION

We have found no significant relation between parents' smoking and respiratory symptoms or lung function in their children. Previous reports that "passive smoking" may cause respiratory disease in children (1, 3, 4) were based on symptoms only. They did not exclude the possibility that the effect of passive smoking may be indirect, by transmission of infection from coughing parents to their children. In two studies (6, 7), the association between parental smoking and symptoms could be attributed to increased cough or phlegm among smoking parents. In only one study (2), infant respiratory disease was associated with parental smoking, even if the parents had no symptoms, but this association was absent in children aged 1–5.

In relating parents' and children's symptoms, bias may be introduced when parents answer the questions for their offspring. In our study, all children them-

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TABLE 9

Contribution of parents' residual lung function and smoking habits and family history of asthma to children's lung function

Lung function	R_1^2* (%)	R_2^2† (%)	Significant parent terms			
			Mothers' residual	Fathers' residual	Only mother smokes‡	Family asthma
Boys (n = 353)						
FVC	90.2	90.7	p <.01	p <.01		
$FEV_{1.0}$	89.8	90.3		p <.01		p <.05
PEF	80.6	81.2		p <.01		
MEF50%	68.7	70.8	p <.01	p <.01		
MEF25%	54.4	58.2	p <.01			p <.05
Girls (n = 277)						
FVC	86.2	87.1	p <.05	p <.01		
$FEV_{1.0}$	86.5	87.7	p <.01	p <.01		
PEF	66.4	67.8		p <.01		
MEF50%	53.1	54.8		p <.01	p <.05	
MEF25%	37.4	37.4				

* R_1^2 is the percentage of the variance in children's lung function which is explained by regression on height, weight and age terms.

† R_2^2 is the percentage of the variance in children's lung function which is explained by regression on height, weight, and age terms and on significant parents' residuals, parents' smoking category terms, and family history of asthma term.

‡ Variable reflecting families in which the mother smokes but the father does not. Variables which were never significant reflected other family smoking categories (neither parent smokes, only father smokes, both parents smoke).

selves answered questions about cough and phlegm; there was no significant association between the prevalence of those symptoms in parents and children (table 2). Nor did the children of parents with cough and phlegm have significantly lower lung function. The association between cough and phlegm in parents and children in other studies may result from bias due to parents answering the children's questions.

For wheezing, parents answered the questions for children aged 15 and under, but not for older children. The prevalence of wheeze in parents correlated with that in younger children, but not with wheeze in older children. To test the reliability of parents' reports on their children's wheeze, residual lung function of the younger children was examined. For boys the results follow the same pattern as the symptom analysis, with significantly lower residuals in children whose mother or both of whose parents wheeze than in children whose parents do not wheeze (for $FEV_{1.0}$, PEF and MEF50%, $p < 0.01$). However, no differences were observed in the girls. Thus, the association between wheeze in parents and children may be real, at least in boys.

The significant correlations between lung function residuals of parents and their children indicate familial resemblances in lung function which are not due to inherited similarities in height and weight nor to a family history of asthma. In the Tecumseh study (8), Higgins and Keller found significant correlations between family members in their height- and age-adjusted $FEV_{1.0}$ scores. The present study confirms these findings and extends them to other lung function measurements as well.

However, we did not find the small but significant correlations which Higgins and Keller showed between spouses (table 7). This might be because the number of spouses in Tecumseh (1770 pairs) is larger than ours (376 pairs). Secondly, many of the husbands and wives in our study were

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not examined at the same time; in Tecumseh, spouses were usually seen together, and thus calibration and other factors (e.g., acute respiratory illness in family) might yield slightly higher correlations. In addition, we found no significant correlations between brothers aged 7–17, although there were significant correlations between sisters 7–14 and in brother-sister pairs. Excluding boys aged 15–17, thus making the age grouping comparable to that for girls, does not change the results for brother pairs. Nor are the results different when boys who have smoked cigarettes are excluded. The lack of a significant correlation in brothers is difficult to explain.

After accounting for height, weight, age, sex and race, the additional contribution to children's lung function from the parents, although significant, is relatively small (table 9). It could easily be missed when smaller numbers are studied (9). The largest relative increase of R^2 occurs for MEF25% in boys, where the height, weight and age terms explain 54.5 per cent of the variance; the other family factors add a further 3.8 per cent. Even here, the effect on the absolute value of lung function is small. Predicted MEF25% for 11-year-old boys (height 161 cm, weight 45 kg) is 1.62 liters/sec if only height, weight and age are considered. Adding the average mothers' residual decreases this value to 1.60 liters/sec, and further addition of the family history of asthma variable decreases MEF25% to 1.48 liters/sec. Thus familial factors, apart from those related to height, weight, age, sex and race, are relatively unimportant in determining lung function.

Chronic obstructive lung disease and even more, asthma, may in part be determined genetically (14–16). Children with a family history of asthma have lower residuals for all lung function measurements than children without such a history (table 6). Girls show the same trends as boys but the differences are not so marked. Wheezing in parents is associated with lower lung function residuals in boys but not in girls. Asthma may begin at an earlier age in boys than in girls (17, 18), and boys might be more exposed to antigenic agents in their work or hobbies. Both familial and environmental factors may contribute to reduction of lung function in children who have a family history of asthma or whose parents wheeze.

There have been no convincing reports on the influence of passive smoking on either adults' or children's lung function. In our study, as expected, adult smokers had much lower lung function residuals than the nonsmokers (table 5), but there was no evidence that exposure to one's spouse's cigarette smoke affected lung function. Nor do the results of this study suggest that parents' smoking has anything but a minor effect on children's lung function. The only significant finding was that daughters' flow rates were lower in families in which the mother smoked and the father was a nonsmoker (table 9). This could be due to chance, and the effect is in any case small.

Heavy exposure to other people's smoke causes nonsmokers to absorb carbon monoxide and nicotine and sensitive individuals to develop cough, wheeze, and respiratory distress (19). People are, however, rarely if ever subjected to such extreme conditions in the home. Although an environmental study of 20 homes showed higher particulate levels in homes with smokers, there was no evidence that this was related to the presence or absence of disease in children (20). We conclude that exposure to low levels of smoke produced by cigarette smokers does not result in chronic respiratory symptoms or loss of lung function among children nor among adults.

References

1. Harlap S, Davies AM. Infant admissions to hospital and maternal smoking. Lancet 1:529–532, 1974
2. Colley JRT, Holland WW, Corkhill RT. Influ-
ence of pa
pneumoni
Lancet 2
3. Love GJ,
tive surv
unteer fa
report fr
Protection
650/1-74-0
4. Cameron
ronment t
Psychol 5
5. Editorial
children's
6. Colley JR
and paren
Br Med J
7. Lebowitz
toms relat
Chest 69
8. Higgins
chronic re
blance in
28:239–25
9. Cotes JE,
nants of
twins. Sym
Human Bi
its Genetic
Taylor and
10. Lerdin S,
Influence

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Source: https://www.industrydocuments.ucsf.edu/docs/jpyx0000

ence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. Lancet 2:1031-1034, 1974
3. Love GJ, Cohen AA, Finklea JF, et al. Prospective surveys of acute respiratory disease in volunteer families: 1970-71 New York Studies. A report from CHESS 1970-71. Environmental Protection Agency: Publication number EPA-650/1-74-004, 1974
4. Cameron P, Robertson D: Effect of home environment tobacco smoke on family health. J Appl Psychol 57:142-147, 1973
5. Editorial: Warning: smoking may damage your children's health. Br Med J 1:1179-1180, 1977
6. Colley JRT: Respiratory symptoms in children and parental smoking and phlegm production. Br Med J 2:201-204, 1974
7. Lebowitz MD, Burrows B: Respiratory symptoms related to smoking habits of family adults. Chest 69:48-50, 1976
8. Higgins M, Keller J: Familial occurrence of chronic respiratory disease and familial resemblance in ventilatory capacity. J Chronic Dis 28:239-251, 1975
9. Cotes JE, Heywood C, Laurence KM. Determinants of respiratory function in boy and girl twins. Symposium of the Society for the Study of Human Biology on Physiological Variation and its Genetic Basis. Edited by JS Weiner. London, Taylor and Francis (in press)
10. Leeder SR, Corkhill RT, Wysocki MJ, et al: Influence of personal and family factors on ventilatory function of children. Brit J Prev Soc Med 30:219-224, 1976
11. Mitchell CA, Schilling RSF, Bouhuys A: Community studies of lung disease in Connecticut: Organization and methods. Am J Epidemiol 103:212-225, 1976
12. Schoenberg JB, Beck GJ, Bouhuys A: Growth and decay of pulmonary function in healthy blacks and whites (Manuscript)
13. US Department of Labor Directory of Occupational Titles. Vols I & II: 3rd edition. Washington, DC, US GPO, 1965
14. Schwartz M: Heredity in bronchial asthma Acta Allerg 5: suppl 2, 1952
15. Lubs MLE: Empiric risks for genetic counseling in families with allergy. J Pediatr 80:26-31, 1972
16. Larson RK, Barman ML, Keuppers F, et al: Genetic and environmental determinants of chronic obstructive pulmonary disease. Ann Intern Med 72:627-632, 1970
17. Broder I, Higgins MW, Mathews KP, et al: Epidemiology of asthma and allergic rhinitis in a total community, Tecumseh, Michigan. J Allergy Clin Immunol 53:127-136, 1974
18. Davis JB. Asthma and wheezy bronchitis in children. Clin Allergy 6:329-338, 1976
19. Editorial. Tobacco smoke and the non-smoker. Lancet 1:1201-1202, 1974
20. Binder RE, Mitchell CA, Husein HR, et al: Importance of the indoor environment in air pollution exposure. Arch Environ Health 31:277-279, 1976

2023383097

Source: https://www.industrydocuments.ucsf.edu/docs/jpyx0000